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~~Art~~ RC 681 A1 C5

From: Qian, Celine
Sent: Wednesday, February 25, 2004 7:04 PM
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Subject: ILL_Order

Please make copy of the following articles. Thank you.

Percutaneous electromechanical mapping demonstrates efficacy of pVGI.1 (VEGF2) in an animal model of chronic myocardial ischemia.
AU Vale, Peter R. [Reprint author]; Tkebuchava, Tengis [Reprint author]; Milliken, Charles E. [Reprint author]; Chen, Donghui [Reprint author]; Symes, James F. [Reprint author]; Isner, Jeffrey M. [Reprint author]
CS St Elizabeth's Med Ctr, Boston, MA, USA
SO Circulation, (Nov. 2, 1999) Vol. 100, No. 18 SUPPL., pp. I.22. print. #109
Meeting Info.: 72nd Scientific Sessions of the American Heart Association. Atlanta, Georgia, USA. November 7-10, 1999.

Celine Qian
Art Unit 1636
Rensem 2A89
571-272-0777

WEST Search History

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DATE: Wednesday, February 25, 2004

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L3	L2 near5 expression vector	18
☐	L2	VEGF-2 or VEGF-C or VEGF2 or VEGFC or VEGF 2 or VEGF C	834
☐	L1	pVGI.1	2

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 NEWS 6 OCT 10 PCTFULL: Two new display fields added
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 NEWS 10 DEC 08 CABA reloaded with left truncation
 NEWS 11 DEC 08 IMS file names changed
 NEWS 12 DEC 09 Experimental property data collected by CAS now available
          in REGISTRY
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 NEWS 15 DEC 18 BIOTECHNO no longer updated
 NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer
          available
 NEWS 17 DEC 22 Additional INPI reactions and pre-1907 documents added to
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          databases
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fields
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          and searchable
 NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in
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 NEWS 22 FEB 05 German (DE) application and patent publication number format
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 NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00,
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=> s pVGI.1
L1          2 PVGI.1

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          2 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L2  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
AN  2002:122826  CAPLUS
DN  136:178364
TI  Vascular endothelial growth factor 2 nucleic acids, polypeptides and
    polypeptide fragments for use in treating various disease states
IN  Coleman, Timothy A.
PA  Human Genome Sciences, Inc., USA
SO  PCT Int. Appl., 241 pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.      KIND  DATE        APPLICATION NO.  DATE
    --------------- ----  ----------  ---------------  --------
PI  WO 2002011769   A1    20020214    WO 2001-US24658  20010803
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
           CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM,
           HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS,
           LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO,
           RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ,
           VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
        RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
           DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
           BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
    AU 2001084734   A5    20020218    AU 2001-84734    20010803
    EP 1313512      A1    20030528    EP 2001-963814   20010803
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
           IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
    US 2003215921   A1    20031120    US 2001-921143   20010803
    NZ 518077       A     20031128    NZ 2001-518077   20010803
PRAI US 2000-223276P  P   20000804
    WO 2001-US24658   W   20010803
AB  Disclosed are human VEGF-2 polypeptides, biol. active, diagnostically or
    therapeutically useful fragments, analogs, or derivs. thereof, and DNA
    (RNA) encoding such VEGF-2 polypeptides. Also provided are procedures for
    producing such polypeptides by recombinant techniques and antibodies and
    antagonists against such polypeptides. Such polypeptides and
    polynucleotides may be used therapeutically for stimulating wound healing
    and for vascular tissue repair. Also provided are methods of using the
    antibodies and antagonists to inhibit tumor angiogenesis and thus tumor
    growth, inflammation, diabetic retinopathy, rheumatoid arthritis, and
    psoriasis.
RE.CNT 7    THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
RECORD
          ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2  ANSWER 2 OF 2  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  2000:30458  BIOSIS
DN  PREV200000030458
TI  Percutaneous electromechanical mapping demonstrates efficacy of
     ***pVGI*** . ***1***  (VEGF2) in an animal model of chronic myocardial
    ischemia.
AU  Vale, Peter R. [Reprint author]; Tkebuchava, Tengis [Reprint author];
    Milliken, Charles E. [Reprint author]; Chen, Donghui [Reprint author];
    Symes, James F. [Reprint author]; Isner, Jeffrey M. [Reprint author]
CS  St Elizabeth's Med Ctr, Boston, MA, USA
SO  Circulation, (Nov. 2, 1999) Vol. 100, No. 18 SUPPL., pp. I-22. print.
    Meeting Info.: 72nd Scientific Sessions of the American Heart Association.
    Atlanta, Georgia, USA. November 7-10, 1999.
    CODEN: CIRCAZ. ISSN: 0009-7322.
DT  Conference; (Meeting)
    Conference; Abstract; (Meeting Abstract)
LA  English
ED  Entered STN: 13 Jan 2000
    Last Updated on STN: 31 Dec 2001


=> s VEGF2 or VEGF 2 or VEGFC or VEGF C
L3       1125 VEGF2 OR VEGF 2 OR VEGFC OR VEGF C

=> s l3 and expression vector
L4         11 L3 AND EXPRESSION VECTOR

=> dup rem l4\
ENTER L# LIST OR (END):l4
'L4\' IS NOT VALID.  VALID FILE NAMES ARE 'BIOSIS, EMBASE, CAPLUS'
You have entered a file name of duplicates to keep that is not
referenced by any of the L#s specified for this DUPLICATE command.
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Please enter one of these file names.

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5          9 DUP REM L4 (2 DUPLICATES REMOVED)

=> s l5 not l1
L6          8 L5 NOT L1

=> d bib abs 1-
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YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:441586 BIOSIS
DN PREV200200441586
TI Modulation of host gene expression by the constitutively active G protein-coupled receptor of Kaposi's sarcoma-associated herpesvirus.
AU Polson, Andrew G.; Wang, David; DeRisi, Joseph; Ganem, Don [Reprint author]
CS Department of Microbiology and Immunology, 513 Parnassus Avenue, Box 0414,
San Francisco, CA, 94143, USA
ganem@cgl.ucsf.edu
SO Cancer Research, (August 1, 2002) Vol. 62, No. 15, pp. 4525-4530. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 21 Aug 2002
Last Updated on STN: 21 Aug 2002
AB Kaposi's sarcoma-associated herpes virus (KSHV) infects B cells and microvascular endothelium, and is linked to both lymphoid and endothelial neoplasms. KSHV encodes a G protein-coupled receptor (v-GPCR) that can bind several CC and CXC chemokines but is able to signal in the absence of known ligands. This signaling can transform cultured fibroblasts, promote angiogenesis in vitro and in vivo, and activate the mitogen-activated protein kinase, c-Jun-NH2-terminal kinase, and p38 pathways. To assess the potential impact of v-GPCR signaling on host cell biology we have examined cellular gene expression in v-GPCR-transfected cells using DNA microarrays. v-GPCR expression up-regulated numerous cellular transcripts in both BJAB B cells and SLK endothelial cells, but with a remarkable degree of cell-type specificity. Among the most highly regulated genes in endothelial cells were the cytokines interleukin 6 and GROalpha; several genes affecting endothelial/vascular growth and remodeling were also induced, including plasminogen, thrombomodulin, the urokinase-type plasminogen activator receptor, and to a modest extent vascular endothelial growth factor C. By contrast, the most highly regulated genes in B cells were the CC chemokines macrophage inflammatory protein 1alpha and macrophage inflammatory protein 1beta. No genes other than members of the dual-specificity phosphatase family were induced in both cell lines. The results indicate that the effects of KSHV GPCR expression in these two target cell types differ considerably and suggest that signaling by this molecule may make different contributions to the pathogenesis of KSHV-related endothelial and lymphoproliferative lesions.

L6 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:275961 BIOSIS
DN PREV200200275961
TI NogaTM left ventricular mapping to assess percutaneous catheter-based gene transfer of vascular endothelial growth factor 2 (***VEGF*** - ***2***) in a placebo-controlled, double-blind trial of patients with chronic myocardial ischemia.
AU Vale, Peter Richard [Reprint author]; Milliken, Charles E. [Reprint author]; Fortuin, David; Symes, James F.; Schatz, Richard A.; Losordo, Douglas W.
CS St Elizabeth's Med Ctr of Boston, Boston, MA, USA
SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.664. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 8 May 2002
Last Updated on STN: 8 May 2002

L6 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:263364 BIOSIS
DN PREV200200263364
TI Inhibition of endothelial cell surface expression of vascular endothelial growth factor-2 using an intrabody strategy.
AU Wheeler, Yurong Y. [Reprint author]; Kute, Timothy E. [Reprint author]; Willingham, Mark C. [Reprint author]; Chen, Si-Yi; Sane, David C.
CS Wake Forest Univ Sch of Med, Winston-Salem, NC, USA
SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.68. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 1 May 2002
Last Updated on STN: 1 May 2002

L6 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:322890 BIOSIS
DN PREV200100322890
TI Human cutaneous fatty acid-binding protein induces metastasis by up-regulating the expression of vascular endothelial growth factor gene in rat Rama 37 model cells.
AU Jing, Chun; Beesley, Carol; Foster, Christopher S.; Chen, Haijuan; Rudland, Philip S.; West, David C.; Fujii, Hiroshi; Smith, Paul H.; Ke, Youqiang [Reprint author]
CS Molecular Pathology Laboratory, Department of Pathology, University of Liverpool, Liverpool, L69 3BX, UK
yqk@liv.ac.uk
SO Cancer Research, (June 1, 2001) Vol. 61, No. 11, pp. 4357-4364. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 4 Jul 2001
Last Updated on STN: 19 Feb 2002
AB Human cutaneous fatty acid-binding protein (C-FABP) gene is capable of inducing the metastatic phenotype when overexpressed in nonmetastatic rat Rama 37 cells. However, the mechanism of how it induces metastasis is not clear. Northern and slot blot analyses revealed that expression of the endogenous vascular endothelial growth factor (VEGF) gene was increased by 3.8-5.2-fold in the C-FABP-transfected cells (pSV-CFABP-R37) and in their metastatic sublines (e.g., Met-1) when compared with that in the nonmetastatic control transfectant pSV-R37 cells generated by transfection of only plasmid DNA. Higher levels of VEGF immunoreactive protein were also secreted from the malignant C-FABP-expressing cells. Reverse transcription-PCR detected two VEGF transcript isoforms, VEGF164 and VEGF188, in both the nonmetastatic control transfectant pSV-R37 cells and the malignant metastatic Met-1 cells. Chick chorioallantoic membrane assays showed that the conditioned medium of the control pSV-R37 cells possessed only very weak angiogenic activity, whereas conditioned media from the metastatic C-FABP transfectants and their sublines were strongly angiogenic and could be inhibited by antibodies to VEGF. Transfection of VEGF164 cDNA in an ***expression*** ***vector*** into nonmetastatic Rama 37 cells produced a cell clone (R37- ***VEGF*** - ***2***) that expressed high levels of VEGF. Inoculation of R37- ***VEGF*** - ***2*** cells into syngeneic Wistar Furth rats produced metastases in a significant number (Fisher's exact test, P<0.01) of animals (18 of 31 animals), whereas the control, vector alone-transfected R37-PSV cells produced no metastases (0 of 30 animals). Immunocytochemical methods demonstrated a strong positive staining for VEGF and an increased microvessel density in the primary tumors produced from PSV- ***VEGF*** - ***2*** cells in comparison with tumors produced from control transfectants. Immunocytochemical staining for factor VIII detected a 3.5-fold increase in microvessel density of the primary tumors produced by PSV- ***VEGF*** - ***2*** cells when compared with that of the primary tumors developed from the control pSV-R37 cells. Therefore, we suggest that overexpression of the C-FABP gene in the original transfectants induces metastasis through up-regulation of expression of the VEGF gene in this rat Rama 37 model system, and thus VEGF may play a crucial role in this particular metastatic cascade.

L6 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:59111 BIOSIS
DN PREV200100059111
TI Active interaction of human A375 melanoma cells with the lymphatics in vivo.
AU Papoutsi, Maria; Siemeister, Gerhard; Weindel, Karin; Tomarev, Stanislav I.; Kurz, Haymo; Schaechtele, Christoph; Martiny-Baron, Georg; Christ, Bodo; Marme, Dieter; Wilting, Joerg [Reprint author]
CS Lehrstuhl II, Anatomisches Institut der Albert-Ludwigs-Universitaet, Albertstrasse 17, 79104, Freiburg im Bresgau, Germany
wilting@uni-freiburg.de
SO Histochemistry and Cell Biology, (November, 2000) Vol. 114, No. 5, pp. 373-385. print.
ISSN: 0948-6143.
DT Article
LA English
ED Entered STN: 24 Jan 2001
Last Updated on STN: 12 Feb 2002
AB We have used the avian chorioallantoic membrane (CAM) to study the interaction of tumor cells with the lymphatics in vivo. The vascular endothelial growth factor-C (***VEGF*** - ***C***) has been shown to be lymphangiagenic. We have therefore grown ***VEGF*** - ***C*** -expressing human A375 melanoma cells on the CAM. These tumors induced numerous lymphatics at the invasive front, and compressed or destroyed VEGF receptor (R)-3-positive lymphatics were observed within the solid tumors. The lymphatics in the CAM and in the A375 melanomas could also be demonstrated with an antibody against Prox 1, a highly specific marker of lymphatic endothelial cells. Proliferation studies revealed a BrdU labeling index of 11.6% of the lymphatic endothelial cells in the tumors and at their margins. A great number of melanoma cells invaded the lymphatics. Such interactions were not observed with ***VEGF*** - ***C*** -negative Malme 3 M melanoma cells. Lymphangiogenesis was inhibited to some extent when A375 melanoma cells were transfected with cDNA encoding soluble VEGFR-3 (sflt4), and the BrdU labeling index of the lymphatics in these tumors was 3.9%. Invasion of lymphatics and growth of blood vascular capillaries were not inhibited by the transfection. Therefore, tumor-induced lymphangiogenesis seems to be dependent to some extent on ***VEGF*** - ***C*** /flt4 interactions, but invasion of lymphatics seems to be a distinct mechanism.

L6 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2004:45996 CAPLUS
TI Construction of eukaryotic ***expression*** ***vector*** for human vascular endothelial growth factor C gene
AU Gao, Jie; Liu, Zhiyu; Dong, Ping; Bi, Yushun; Tian, Hua; Li, Guibao; Song, Tao
CS Department of Anatomy, School of Medicine, Shandong University, Peop. Rep. China
SO Shandong Daxue Xuebao, Yixueban (2003), 41(2), 151-154
CODEN: SDXYBZ; ISSN: 1671-7554
PB Shandong Daxue Xuebao, Yixueban Bianjibu
DT Journal
LA Chinese
AB The eukaryotic ***expression*** ***vector*** for human vascular endothelial growth factor C (***VEGF*** ***C***) gene was constructed for further study on the role of ***VEGF*** - ***C*** gene in lymph angiogenesis. According to human ***VEGF*** - ***C*** cDNA sequence, a pair of specific primers contg. digestion site of EcoR I and BamH I on the 5 end were designed and constructed, then reverse transcription polymerase chain reaction (RT-PCR) was used to clone ***VEGF*** - ***C*** cDNA from human breast cancer cell MDA-MB-231. After being purified, the product of RT-PCR (1.28 Kb) was ligated into a clone vector pMD18-T. The recombinant plasmid pMD18-T, first propagated in Escherichia coli DH5.alpha., then extd. purified and digested with EcoR I and BamH I, was confirmed to contain full-length ***VEGF*** - ***C*** cDNA by agarose gel anal. and DNA sequence anal. The resultant EcoR I-BamH I fragment (1.27 Kb) which contained the full-length human ***VEGF*** - ***C*** cDNA was ligated into eukaryotic ***expression*** ***vector*** pcDNA3.1(-) digested with EcoR I and BamH I. The pcDNA3.1(-)/ ***VEGF*** - ***C*** , digested with EcoR I and BamH I, contained the ***VEGF*** - ***C*** cDNA sequence identified by agarose gel electrophoresis. The pcDNA3.1(-)/ ***VEGF*** - ***C*** , a eukaryotic ***expression*** ***vector*** for human ***VEGF*** - ***C*** , was constructed successfully.

L6 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:265459 CAPLUS
DN 134:290751
TI Recombinant single-chain receptor antagonist proteins and their use in treatment of inflammatory disorders
IN Halkier, Torben; Schambye, Hans Thalsgard; Okkels, Jens Sigurd; Andersen, Kim Vilbour; Nissen, Torben Lauesgaard; Soni, Bobby; Jeppesen, Claus Bekker; Van Den Hazel, Bart
PA Maxygen Aps, Den.
SO PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001025277	A1	20010412	WO 2000-DK563	20001006
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1226173	A1	20020731	EP 2000-965860	20001006
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
US 2004014948	A1	20040122	US 2003-444691	20030523
PRAI DK 1999-1438	A	19991007		
DK 1999-1855	A	19991223		
DK 2000-1119	A	20000720		
US 1999-160820P	P	19991021		
US 2000-174655P	P	20000106		
US 2000-225723P	P	20000816		
US 2000-684720	B1	20001006		
WO 2000-DK563	W	20001006		

AB The invention relates to a single-chain oligomeric protein antagonist which binds to an extracellular ligand-binding domain of a cellular receptor of a type requiring binding of an oligomeric ligand to two or more receptor subunits to be activated, the protein comprising at least two, typically structurally homologous, receptor-binding sites of which at least one is capable of binding to a ligand-binding domain of the cellular receptor and at least one is incapable of effectively binding to a ligand-binding domain of the cellular receptor, whereby the single-chain oligomeric protein is capable of binding to the receptor, but incapable of activating the receptor; as well as to nucleotide sequences encoding such single-chain oligomeric proteins, expression vectors comprising such a nucleotide sequence, recombinant host cells comprising such a nucleotide sequence or ***expression*** ***vector*** , methods for producing the nucleotide sequences and proteins, pharmaceutical compns. comprising the single-chain oligomeric protein, and use of the single-chain oligomeric protein for the prodn. of medicaments and in therapy. A preferred single-chain antagonist according to the invention is a TNF-.alpha. antagonist. Thus, a single-chain TNF-.alpha. protein comprising of 3 human TNF-.alpha. chains connected by linker peptides was produced with Saccharomyces cerevisiae and shown to be an agonist of the TNF-.alpha. receptor. The same TNF-.alpha. trimer contg. Y87R mutations in the first and third copies of TNF-.alpha. was also prepd. This was shown to be a partial TNF-.alpha. agonist and a competitive antagonist of the TNF-.alpha. receptor.
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:46307 CAPLUS
DN 128:152421
TI Down-regulation of vascular endothelial growth factor in a human colon carcinoma cell line transfected with an antisense ***expression*** ***vector*** specific for c-src
AU Ellis, Lee M.; Staley, Charles A.; Liu, Wenbiao; Fleming, R. Y. Declan; Parikh, Nila U.; Bucana, Corazon D.; Gallick, Gary E.
CS Departments Surgical Oncology, Cell Biology, Univ. Texas M.D. Anderson Cancer Center, Houston, TX, 77030, USA
SO Journal of Biological Chemistry (1998), 273(2), 1052-1057
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Vascular endothelial growth factor (VEGF) is implicated in the angiogenesis of human colon cancer. Recent evidence suggests that factors that regulate VEGF expression may partially depend on c-src-mediated signal transduction pathways. The tyrosine kinase activity of Src is activated in most colon tumors and cell lines. The authors established stable subclones of the human colon adenocarcinoma cell line HT29 in which Src expression and activity are decreased specifically as a result of a transfected antisense ***expression*** ***vector*** . This study detd. whether VEGF expression is decreased in these cell lines and whether the smaller size and reduced growth rate of antisense vector-transfected cell lines in vivo might result, in part, from reduced vascularization of tumors. Northern blot anal. of these cell lines revealed that VEGF mRNA expression was decreased in proportion to the decrease in Src kinase activity. Under hypoxic conditions, cells with decreased Src activity had a <2-fold increase in VEGF expression, whereas parental cells had a >50-fold increase. VEGF protein in the supernatants of cells was also reduced in antisense transfectants compared with that from parental cells. In nude mice, s.c. tumors from antisense transfectants showed a significant redn. in vascularity. Apparently, Src activity regulates the expression of VEGF in colon tumor cells.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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